UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON D.C. 20460

OFFICE OF THE ADMINISTRATOR
SCIENCE ADVISORY BOARD

July 11, 2008

EPA-COUNCIL-08-001

The Honorable Stephen L. Johnson
Administrator
U.S. Environmental Protection Agency
1200 Pennsylvania Avenue, N.W.
Washington, D.C. 20460

Subject: Benefits of Reducing Benzene Emissions in Houston, 1990-2020

Dear Administrator Johnson:

The Advisory Council on Clean Air Compliance Analysis (Council) was asked to review a recent case study on the benefits of reducing benzene emissions. The "Air Toxics Case Study – Health Benefits of Benzene Reductions in Houston, 1990-2020" was conducted by EPA's Office of Air and Radiation (OAR) as part of its "812 Study," an ongoing comprehensive analysis of the total costs and total benefits of Clean Air Act Amendment (CAAA) programs. As recognized by OAR, the challenges for assessing progress in health improvement as a result of reductions in emissions of hazardous air pollutants (HAPs) are daunting. Accordingly, EPA has been unable to adequately assess the economic benefits associated with health improvements from HAP reductions due to a lack of exposure-response functions, uncertainties in emissions inventories and background levels, the difficulty of extrapolating risk estimates to low doses and the challenges of tracking health progress for diseases, such as cancer, that have long latency periods. Benzene, however, has a large epidemiological database which OAR used to estimate the health benefits of benzene reductions due to CAAA controls. The Council was asked to consider whether this case study provides a basis for determining the value of such an exercise for HAP benefits characterization nationwide. The Council, augmented with members of its Health Effects Subcommittee, provides detailed advice in the enclosed Advisory with highlights summarized below.

The benzene case study estimated the health benefits from an average reduction in benzene concentrations of 1 $\mu g/m^3$ in the Houston area. Those health benefits were estimated as 9 avoided leukemia cases in the Houston area during the study period (1990 through 2020). Although uncertainties and omitted factors could raise or lower this estimate, we believe it is more likely to underestimate the health benefits. First, the study did not incorporate epidemiological data pointing to associations between benzene and other types of cancers. Second, because of the latency period associated with cancer, the health benefits of reduced benzene emissions through the 2020 terminus for the study

would extend beyond 2020. Third, the case study did not include the impact of the Mobile Source Air Toxics rule promulgated in 2006. Fourth, additional health benefits accruing to individuals living in homes with attached garages were not included in the base estimate. (OAR acknowledged these limitations in the case study.) Fifth, the estimate did not incorporate an integrated approach for projections of population change, economic activity, or emissions. Nonetheless, the Council lauds the OAR's air quality and exposure modeling, the life table approach for estimating health benefits and supplemental analyses of individuals in high-exposure environments. Given these achievements, the case study offers a reasonable, if qualified, estimate of health benefits.

For future studies, the Council urges greater attention to the discrepancies between emissions inventories and the reality of monitored concentrations and to a richer discussion of the relative importance of sources of uncertainty. Recent air quality studies for southeast Texas indicate that industrial inventories underestimate actual emissions by a factor of two or more for some hydrocarbons. These inventory under-predictions have been attributed to missing sources and under-prediction of inventoried sources. Since there is a systemic under-prediction of observed benzene concentrations in this case study, by a factor of two or more in some locations, it is possible that the inventory used in this work significantly under-estimated emissions. We do not know whether reductions under CAAA would apply to the missing sources, but we urge greater attention to this issue, given that accurate emissions inventories are a critical need for a plethora of policy purposes.

The benzene case study successfully synthesized best practices and implemented the standard damage function approach to estimating the benefits of reduced benzene, however the Council is not optimistic that the approach can be repeated on a national scale or extended to many of the other 187 air toxics due to insufficient epidemiological data. With some exceptions, it is not likely that the other 187 HAPs will have the quantitative exposure-response data needed for such analysis. Given EPA's limited resources to evaluate a large number of HAPs individually, the Council urges EPA to consider alternative approaches to estimate the benefits of air toxics regulations. We underscore the National Research Council's call for more integrated multi-pollutant approaches (National Research Council, *Air Quality Management in the United States*, 2004). One example of such an approach is OAR's Risk and Technology Review (RTR) program that seeks to evaluate air toxics risk by source category. Economies of scale may also be found in the emerging 3D air quality modeling work that can now include individual air toxics so that HAPs need not be modeled separately.

Detailed recommendations are included in the enclosed Advisory. On behalf of the entire Council, we appreciate this opportunity to provide timely advice to the Agency. We hope these comments are helpful to the Office of Air and Radiation as it proceeds with this important work.

Sincerely,

/Signed/

James K. Hammitt, Chair
Advisory Council on Clean Air Compliance Analysis

Enclosures

U.S. Environmental Protection Agency
Advisory Council on Clean Air Compliance Analysis
Review of the Benzene Air Toxics Health Benefits Case Study

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Advisory Council on Clean Air Compliance Analysis
Advisory on Health Benefits of Benzene Reductions in Houston, 1990-2020

1. ***<u>General Review</u>. Please comment on the validity, reliability and utility of this case study and whether it achieved its purpose in contributing to the science of estimating the benefits of reduced concentrations of air toxics. Specifically, please comment on EPA's data choices and characterization of results given these data choices; EPA's methodological choices made for analyzing the data; and implications this case study may have for future analyses.***

Overall, the Council concludes that this case study is of high quality and is in large part clearly documented. It provides a reasonably comprehensive estimate of the primary health benefits of reductions in benzene emissions in the Houston area associated with regulations enacted pursuant to the 1990 Clean Air Act Amendments.

The case study uses a damage-function approach that follows a linear causal chain: regulations induce reductions in benzene emissions that lead to lower ambient concentrations and reduced human exposure to benzene, which leads to smaller cancer risk to Houston-area residents, a welfare improvement that can be quantified in monetary terms. A strength of this approach is that it follows a logical causal chain and that relevant data (notably emission inventories and estimates of the relationship between exposure and health effects based on human-epidemiological data) are available to characterize each link in the chain. (Indeed, benzene was selected for the case study because of the availability of relevant data.) This approach is more difficult to apply to pollutants for which exposure-response functions are less-well suited to estimating the probability of health effects, either because the function must be extrapolated from animal data (in which case the function may be intentionally biased upward so as to yield an overestimate of risk) or because there are only estimates of reference concentrations (or other possible thresholds) that provide little information on how the probability of a health response changes with respect to changes in exposure.

The case study relies on detailed, fine-scale modeling to estimate changes in ambient concentrations and human exposure to benzene. This fine-scale modeling is valuable for pollutants that exhibit sharp spatial and temporal gradients, but is resource-intensive. It is not clear that a national-scale assessment of the benefits of benzene regulation using this detailed modeling would be cost-justified, much less the national scale benefits of detailed modeling for many of the other 187 air toxics identified by Congress in 1990. The Council encourages EPA to consider how to use a less intensive approach to obtain an alternative estimate of the benefits of benzene regulation in the Houston area and to compare the results of the simpler approach with the results reported in the current case study. (One possible approach would be to use a reduced-form re-analysis using National Air Toxics Assessment concentration data, as suggested in the last paragraph of the Executive Summary.)

The Council notes several limitations of the case study. First (unlike the main Section 812 analysis), the case study does not rely on an integrated projection of future population, economic activity, and emissions. In the case study, economic activity (and with it, benzene emissions in the without-CAAA case) is projected to increase substantially, but the population is held constant at its 2000 level and distribution. Similarly, vehicle miles traveled (VMT) are projected to grow but with no change in road infrastructure. Moreover, there is no feedback between regulation, air quality and behavior. For example, regulations that increase vehicle costs could slow the rate of introduction of new vehicles (increasing emissions); improved air quality could alter residential patterns and the timing and location of outdoor recreational and other activities (and hence exposure). Many of these effects are likely to be quantitatively unimportant, but the Council suggests that EPA consider which, if any, may be important and discuss them.

Second, the Council suggests it would be more useful to report the expected number and monetary value of averted fatal and non-fatal leukemia cases by the year in which they would have arisen, rather than reporting only the cumulative totals that grow over time simply because health benefits are incurred each year (see, e.g., Table ES-1). Averted cases are more appropriately conceived as a flow rather than a stock. From this perspective, benefits would be reported as "deaths avoided per year or per decade". The fact that the expected number of cases per year may be less than one poses no conceptual difficulty (this fact appears to be one reason EPA presents cumulative rather than annual values). Moreover, the Council suggests that EPA include in its base-case results the cases averted by lower benzene exposures through 2020, even if these cases would have arisen after 2020 (i.e., the cases described in the subsection "Expected Total Benefits" on pp. 3-9 – 3-10). (The Council does not object to reporting totals over the period evaluated as a supplement to the annual values.)

This case was selected in large part because of the availability of relevant data on exposure-response relationships, emissions, and atmospheric concentrations. The case study serves as proof-of-concept that benefits of CAAA regulations can be reasonably quantified for at least one air toxic and one location, but the approach clearly cannot be repeated with this level of detail for many of the other 187 toxics, or even for benzene at national scale. The Council encourages EPA to consider what simpler approaches could be used to estimate the benefits of air-toxic regulation and to use the results of this case study as one point of comparison for evaluating the results of simpler approaches.

2. ***Emissions Estimations. EPA developed a benzene emissions inventory for the three counties in the case study, based on EPA's National Emissions Inventory (NEI), MOBILE6.2 model, and NONROAD 2004 model. Please comment on this approach to emissions estimation.***

EPA used current best practices and approaches to estimating emissions for this case study. The resulting inventory indicates that in the base year, industrial sources of benzene are substantial. As the with- and without-CAAA scenarios are applied,

substantial reductions in benzene emissions occur in all source categories, but the reductions are especially large in the industrial sector.

Because industrial emissions are a significant fraction of both the base case inventory and the projected emission reductions, it is important to characterize the uncertainties in these emissions. Recent air quality studies done in southeast Texas (the first and second Texas Air Quality Studies, TexAQS I and II) indicate that for some hydrocarbon emissions, industrial inventories underestimate actual emissions by a factor of two or more (Ryerson, et al., 2003; Kleiman, et al., 2002; Allen and Durrenberger, 2003). These inventory under-predictions have been attributed to missing sources and under-predictions of inventoried sources.

Observed benzene concentrations are evidently under-predicted on average in this case study, with predicted values less than half the observed value at a significant fraction of monitors (p. 3-7). This under-estimation may reflect underestimates of emissions in the emissions inventory. If so, the effect of this underestimate on estimated benefits of regulation depends on whether the underestimated emission sources are affected by CAAA regulations or not: if they are not affected, failing to include them in the model may have little effect on the change in benzene concentrations and on health benefits; if emissions from these sources are reduced by the CAAA regulations, benefits are likely to be underestimated.

Improvements in the emission inventory are likely to continue, and if future studies continue to employ best current emission inventory practices, the quality of the estimated concentration changes is likely to improve. At present, however, current information and the model performance as compared with observation suggest a systemic under-prediction of benzene emissions. In the report, a summary judgment is offered that missing information and general uncertainty is likely to lead to an under-representation of emissions (p. 4-2).

The goals of the case study include identification of limitations and gaps in the data needed for analysis and provision of an estimate of the uncertainties. There is some effort to discuss uncertainty, but it is not treated in an integrated fashion. The Council encourages a stronger narrative about the relative importance of different sources of uncertainty and some discussion about how uncertainty could be better addressed in future analysis.

It is not clear exactly what requirements of the CAAA of 1990 are included in the analysis. In each subsection of the emissions appendix, these could be specified. For the mobile source section, for example, it is not clear which regulations affect benzene levels and why. The I/M rules, ATP programs, and fuels programs appear to reduce benzene, but the details of how they do so are not clear.

In addition to these general observations, the Council offers some specific comments:

1. Temperature and humidity play a role in emissions from some sources. By 2020, climate may be expected to cause greater emissions on a given projection of economic activity. Is this incorporated in the analysis?
2. A hierarchy of sources for emissions data for the base years 1990 and 2002 is given. In some cases information from these various sources is contrasted, but there is no systematic evaluation of the (dis)harmony of information across these sources.
3. The personal transportation model accounts for turnover of the vehicle fleet. Does this include technological changes that would affect evaporative emissions?
4. It appears that a factor accounts for the entry of new nonroad vehicles into the market by accounting for sales. Does this account for changes in use and retirement of existing vehicles, and for technological differences?
5. Have industrial leak detection and repair reductions, that are part of the Texas State Implementation Plan for attaining the ozone NAAQS, been incorporated into the with-CAAA emission scenarios?
6. Appendix A p. 41: "The differences in the distribution among the top categories could be the result of the [sic] including facilities as point sources in the 1996 NTI inventory that were aggregated and reported as nonpoint sources in the 2002 NEI." This is easily verified. Does the sum of point and non-point emissions from each data source agree?
7. Appendix A pp. 66-68: Are the VMT figures for 2002 and 2009 linked to projections of energy prices in the DOE study used earlier in the appendix? That is, are the assumptions internally consistent? If energy prices increase, VMT should respond. Note that the CAAA may affect gasoline prices (e.g., by requiring reformulated gasoline). Do these differences in gasoline prices lead to higher VMT in the without-CAAA scenarios?
8. Estimating emissions bottom-up from a plant-level inventory seems reasonable, and the detailed discussion of the process in the Pechan report showed that the inventory was carefully constructed. The projections to 2020 based on estimates of industry growth seem less convincing. Given that a few industries are responsible for the bulk of the emissions, it might be worthwhile to pay extra attention to modeling their growth, or considering the impact of different growth assumptions.

3. Air Quality Modeling and Exposure Modeling. EPA used the American Meteorological Society/US EPA Regulatory Model (AERMOD) to estimate changes in ambient concentrations and the Hazardous Air Pollutant Exposure Model (HAPEM6) to estimate individual exposures to benzene levels. Please comment on this approach.

The use of AERMOD to estimate changes in concentrations seems appropriate. While more sophisticated models could be utilized, there are good reasons to use plume-scale models that can resolve individual plumes under conditions when small-scale local sources are important in defining areas of maximum impact. The HAPEM6 model was exercised appropriately and the results appear sound.

However, the relatively large discrepancy between model calculations and monitored benzene concentrations at specific sites suggests some serious deficiencies in either the emissions inventory (discussed in response to question 2) or the AERMOD plume model configuration. The addition of a somewhat arbitrary "background" to the AERMOD calculations using ambiguous methods alleviates only a portion of the discrepancy. There may be a bias introduced in AERMOD by the treatment of calculations during calm periods, and a suggestion is made below to address this shortcoming. However, the Council suspects that calculated differences in concentration due to the CAAA regulations are probably estimated more reasonably than absolute concentrations (i.e., similar errors in estimating absolute concentrations are likely to occur in the with- and without-regulation cases and to roughly offset one another when calculating the difference).

Background concentrations: The method by which "background" concentrations are specified is ambiguous. It appears that some percentile of the cumulative monitored benzene concentrations is used. If background concentrations are added to calculated benzene concentrations when comparing monitored and calculated concentrations, they should be set using monitored concentrations coming from specific wind directions. For example, "background" concentrations probably occur more frequently when winds are coming from a southerly direction (winds blowing from the Gulf of Mexico towards land). Furthermore, the "background" should possibly be wind-direction dependent. That is, when winds are out of the north, monitored concentrations in the northern portion of the domain should be used as a background. Similar procedures could be used for easterly or westerly winds.

Calm periods: There is a probable shortcoming related to using AERMOD during calm periods, which is when high concentrations occur. A significant reason for the serious under-calculation of benzene concentrations may pertain to results from the assumed "steady state" plumes inherent in the AERMOD formulation, coupled with the error of ignoring calm periods. Ignoring calm periods will seriously underweight periods when the greatest concentrations occur, thus resulting in potentially large underestimates of calculated concentrations. Therefore, concentrations must be estimated during calm periods. In earlier EPA plume models, calm winds were set to a minimum value (e.g., 1 m/s), and directions were randomly set or set to the last or next reported non-calm wind direction. These methods of treating calms should be implemented in this study. If 1 m/s winds were assumed during calm periods, significant impacts would be calculated in the vicinity of point sources, especially for surface sources (as opposed to stack emissions) that are presumably significant in the benzene inventory for the Houston area. During calms, emissions build up at the source location and subsequently affect downwind receptors when the wind resumes. Hence the Council recommends that calm periods be modeled by setting wind speed to a minimum value (e.g., 1 m/s, 1.5 m/s) and wind direction be set to the *next* reported wind direction. Thus if measurable winds come from the north for one hour, followed by three hours of calm, then measurable winds from the west, the calm should be modeled as a westerly wind at the selected minimum speed.

Reference meteorology: In a coastal setting such as Houston where land-sea breezes dominate local circulations, the physical location of a meteorological monitor will

strongly influence AERMOD calculations. It is noted that only two surface monitors (IAH and HOU airports) are readily available and the IAH monitor shows fewer missing or calm reports. Missing reports should be used as a primary gauge of monitor quality and monitors or years with the fewest missing observations would be preferred. The Council suggests that there are probably a large number of wind and meteorological observations available in the greater Houston metropolitan area that should be evaluated for use in this study.

The existing study contains a minor ambiguity relating to the choice of the year for which meteorology observations are used as input to the different emission scenarios considered by AERMOD. Different years' meteorology will yield different concentration changes resulting from emission changes. Changes in concentrations between 1990 and 2000 that result only from differences in meteorology are not attributable to CAAA regulations, and should not be highlighted. An additional uncertainty arises since 2010 and 2020 meteorology is not available for simulating concentrations in those years. The Council feels that it is desirable to use as much meteorological information as computationally feasible. Multiple years of meteorology from multiple monitors should be used for all the emission scenarios. Annual average concentrations could then be either averaged or appropriately statistically merged to obtain single values for each receptor. At a minimum, both the 1990 and 2000 meteorology should be used for all emission scenarios and then merged. The calculation of concentration changes for multiple years' of meteorology could potentially provide a basis for describing uncertainty and bounds for sensitivity studies that might be considered in the future.

The analysis should attempt to estimate the expected value (and distribution) of changes in benzene concentrations rather than the realized value in any particular year, which depends on realized meteorological conditions. The years referred to in the study concern the emissions during that year, not the meteorology. For comparing calculated and monitored concentrations, it is appropriate to use meteorological data corresponding to the monitored time period.

4. ***Life table approach for health benefits. Please comment on EPA's life table approach for estimating health benefits, specifically addressing the following:***
    - ***EPA's selection of leukemia as the primary health endpoint;***
    - ***EPA's use of weighted, cumulative exposure measures in the life table risk model to account for the cessation lag in the realization of benefits following benzene exposure reductions;***
    - ***EPA's interpretation of the literature on latency and cessation lag for benzene-induced leukemia;***
    - ***EPA's choice of a linear dose-response function;***
    - ***EPA's sensitivity analyses of the primary benefits estimate (i.e., choice of epidemiological cohort study, the health endpoints of all leukemia versus acute myelogenous leukemia, the lag length, and the exposure values used); and***
    - ***EPA's choice not to apply an adjustment for exposure to benzene in early life.***

The Council commends EPA's use of a life-table approach in this case study. This approach has the potential to more accurately represent the effects of environmental health risks on mortality than the commonly used attributable-death approach that provides little information on the timing of deaths and does not reflect competing mortality risks. The case study shows that it is possible to perform an evidence-based analysis of the health benefits of reduced benzene emissions. The Council suggests that it would be helpful to have a short description of the life-table approach that is accessible to non-specialist readers before reporting details of the analysis.

Leukemia as primary health endpoint: The Council supports the use of all leukemias as the primary health endpoint and sensitivity analyses using AML. We recommend that the criteria used to judge the weight of the evidence be more clearly indicated, perhaps in a table.

Noting that EPA's assessment of the health literature finished in 2005, we suggest that more recent evidence on non-Hodgkins Lymphoma (NHL) and benzene exposure (Smith et al., 2007; Steinmaus et al., 2008) be assessed and, if appropriate, used to calculate risks and benefits that would supplement those for leukemia.

Some studies suggest other health effects, but the evidence is weak at this time. Two topics for possible consideration are:

(i) A Danish study of children's traffic-related exposures, Raaschou-Nielsen et al. (2001), found a near-significant 25% increased risk of lymphomas (p for trend = 0.06) for a doubling of the concentration of benzene during pregnancy.

(ii) A study suggesting that benzene is cardiotoxic and arrhythmogenic. Kotseva and Popov (1998) found that systolic and diastolic blood pressures and the prevalence of arterial hypertension were significantly higher in workers exposed to benzene than in control groups. There was a significant correlation between the length of service and increased systolic and diastolic blood pressure, after controlling for major cardiovascular risk factors. Note that hypertension may predict cardiac mortality, as accepted for lead in the Section 812 retrospective analysis.

Interpretation of the literature on latency and cessation lag: We agree with EPA that the evidence mainly concerns latency, not cessation lag, but that "information about latency can also help inform our estimate for a cessation lag." The Council suggests that EPA provide a clearer explanation about how knowledge of latency can inform judgment about cessation lag, i.e., better exposition of the rationale and model.

The review of evidence on latency, and on the relevant time-window of exposure (summarized in Appendix C, Exhibit 4), appears to be comprehensive. In particular, it is useful to see evidence that the highest risks were associated with relatively recent exposures and that, in general, "distant" exposures had little identifiable effect. We agree with the view expressed in Appendix C of the case study (p.17) that the evidence as

reviewed "points to a lag structure where a new steady-state risk level is reached within 15 years following a regulatory change" and that it is appropriate to incorporate this interpretation into the benefits analysis. We note an additional paper that provides general support for this evaluation: Richardson (2008) found the greatest risks in the 10 years immediately after exposure, a smaller increase in the period 10-19 years after exposure, and no evidence of increased risk at 20 years or more after exposure.

Use of weighted, cumulative exposure measures to account for cessation lag: We believe this method is appropriate for application with the linear dose-response model. However the implications of its use with nonlinear concentration-response models require evaluation.

Choice of a linear dose-response function: We support using a linear exposure-response (E-R) function for the primary analyses and encourage EPA to provide a more complete discussion of the merits of alternative E-R functions and their implications for estimated benefits.

Based on the Pliofilm cohort analyses, a linear (multiplicative) exposure-response function, as used, is reasonable and arguably best. However Crump (1994, 1996) found suggestive though not compelling ($0.05 < p < 0.1$) evidence to support an intensity-dependent quadratic model when using the Paustenbach et al. exposure estimates which Crump (1996) evaluated as the best available at that time. Questions have been raised about the Paustenbach et al. (1992) reanalysis of the Pliofilm cohort data; e.g., the exposure estimates, concurrent exposures, inconsistent assumptions and calculations, estimates of dermal absorption, and lack of control for confounding (e.g., Utterback and Rinksy, 1995). Additionally, Crump's conversion of the Pliofilm data to all age groups added uncertainty to his estimates.

While maintaining a linear (Pliofilm cohort) or supralinear (Chinese cohort) multiplicative model as its first choice, EPA should consider using the quadratic intensity-dependent results for sensitivity analyses – for methodological reasons, because the non-linear model would be more difficult to implement; and for substantive reasons, because Crump (1996, Table 6) suggests it would lead to much lower estimates of the benefits of reducing benzene emissions; i.e., the choice of E-R model is likely to be one of the most influential steps in the benefits analysis.

As noted in EPA's benefits analysis, (Appendix C, pp. 12-13), it is unclear whether lack of a clear effect at low exposures reflects negligible risk or lack of study power (or both). In this context, the use of a no-threshold model for the primary analyses is reasonable and consistent with EPA's Guidelines for Carcinogen Risk Assessment, which favor linear extrapolation to low doses in the face of uncertainty because it is health-protective. Nevertheless, for this analysis, where a best estimate is the goal, EPA should review evidence of a possible threshold and decide whether sensitivity analyses using a threshold model are warranted. For example, recent detailed re-examination by Miller at al. (2005) of three nested case-control studies noted that the case-control study of Glass et al. (2003)

showed clear evidence of increased risks above 16 ppm-years, but not otherwise and found that the other two studies were consistent with this finding.

Sensitivity analyses of the primary benefits estimate: We appreciate that sensitivity analyses were done on several issues where more than one good analytical option was available. These were useful in identifying which issues in practice make a difference to the final estimate of avoided cases.

Choice not to apply an adjustment for exposure to benzene in early life: The Council supports EPA's decision not to apply an adjustment for exposure to benzene in early life. A search of recent literature by one member did not find any new evidence to require inclusion of exposure to benzene in early life.

5. ***Valuation. Please comment on EPA's approach to assigning economic value to avoided cases of leukemia, both fatal and non-fatal, with specific reference to:***
    - ***EPA's use of a "pre-mortality morbidity" supplement to VSL for fatal leukemias;***
    - ***EPA's development of a unit value for a non-fatal case of leukemia based on current literature and previous SAB advice; and***
    - ***EPA's choice not to include a "cancer premium," consistent with the SAB Environmental Economics Advisory Committee (EEAC) panel in 2001***

Overall, EPA has made good choices in valuing the reduced risks of leukemia, both fatal and non-fatal, in the case study.

"Pre-mortality morbidity" supplement: The Council approves of EPA's use of a "pre-mortality morbidity" supplement to VSL for fatal leukemias although it notes that this supplement may be justified for reasons other than the one given. Based on prior SAB guidance, EPA adds an estimate of the medical costs associated with cancer prior to death. This value is interpreted as a cost-of-illness measure of the patient's lost well-being during the period while he/she is suffering from cancer. However, the medical costs are clearly part of the social cost of cancer and so should be included as part of the social value of preventing leukemia, even if there was no private loss in well-being from cancer morbidity. In principle, the component of medical costs born by the individual is incorporated in conventional estimates of VSL, but given the magnitude of medical costs for cancer (estimated as $110,000 in this case), most individuals could not pay them and a large part of these costs must be paid by others (through public or private insurance programs).

Note also that if a large component of medical costs are paid by public sources, the rationale for claiming that these costs underestimate the loss in well-being experienced by the individual collapses. The total loss suffered by an individual includes his private financial costs (e.g., medical expenses and lost income) and the direct utility loss associated with disease. The private cost of illness clearly underestimates this total, as it excludes the utility loss. However, there is no necessary relationship between the value of

publicly paid medical costs and private utility loss – the value of the utility loss to the individual could be much larger or much smaller than the value of the publicly paid medical costs.

This argument implies that it is appropriate to include medical costs in valuing fatal leukemias, but doing so is unlikely to adequately incorporate the loss in well-being associated with pre-mortality morbidity. Improved measures of the value of the pre-mortality morbidity require additional primary research.

Unit value for non-fatal leukemia: The empirical literature on valuation of non-fatal health effects, and specifically non-fatal cancers, is extremely limited. In the absence of more recent estimates using current-practice methods, the Magat et al. (1996) study of risk-risk tradeoffs between non-fatal lymphoma and automobile-accident fatality is cited as a basis for the value of non-fatal lymphoma. One of the limitations of this study is that the latency associated with lymphoma was not specified and so it is uncertain what assumptions respondents made about the timing of lymphoma. In addition to known limitations of the original study, we are concerned about how transferable the value for non-fatal lymphoma is for leukemia. The use of a value for chronic bronchitis as an estimate of the value of non-fatal leukemia clearly lacks face validity.

The Council suggests that EPA provide more information on the duration and severity of non-fatal leukemia, non-fatal lymphoma, and chronic bronchitis for evaluating the possibility of transferring benefit estimates from these other conditions. Severity could be described by symptoms, or perhaps using standard health-related quality of life (HRQL) indexes such as the EuroQoL EQ-5D, Short Form SF-6D, or the Health Utilities Index (HUI). (Some Council members are skeptical of the ability of these generic measures to accurately measure severity, noting that relative severity of different conditions can depend on the index used for measurement.) If the severity and duration of both non-fatal lymphoma and chronic bronchitis are similar to those of non-fatal leukemia, the use of the monetary values for these other conditions would be supported. Similarly, if either non-fatal lymphoma or chronic bronchitis is more similar to the target condition (non-fatal leukemia), more weight could be put on the value of the corresponding health condition. In addition, the Council recommends that the EPA examine the valuation literature for other illnesses with similar severity and duration. Although the EPA appears to believe non-fatal leukemia is a chronic condition, studies of leukemia survivors may indicate that transfers of high-quality acute-condition values are more appropriate.

Cancer premium: VSL estimates derived from wage-risk studies may provide a biased estimate of population willingness to pay for reducing the risk of fatal cancers because of differences in the nature of the risks being valued and characteristics of the affected population. The possibility of environmentally induced cancer may evoke special fears and be seen as a more involuntary, uncontrollable hazard than workplace accidents. Environmentally induced cancers may have a long latency period whereas accident risks are more immediate. In its 2000 guidance, the SAB Environmental Economics Advisory Committee concluded that the empirical literature was insufficient to support the use of an alternative VSL for environmentally induced cancer. The Council does not disagree

with this conclusion, but notes that the relevant literature is growing and encourages EPA to review this issue. Recent relevant studies include: Van Houtven, Sullivan, and Dockins (2008), who find that WTP to reduce cancer risk with a five year latency period is three times larger than WTP to reduce current automobile-accident risks (the cancer premium falls when longer latency periods are considered); Hammitt and Liu (2004), who find weakly significant evidence that WTP to reduce cancer risk is about one-third larger than WTP to reduce risk of an otherwise similar non-cancer disease in a contingent valuation study in Taiwan; and Tsuge, Kishimoto, and Takeuchi (2005), who identify a small but significant preference for avoiding cancer risks in Japan. In addition, Sunstein (2005) argues that VSL figures should be made risk-specific and suggests that the value of a life saved from cancer is at least twice the value of a life saved from a workplace accident.

Studies that have elicited risk-risk and benefit-risk tradeoffs have quantified relative tolerances for different mortality risks. In a study of hormone-replacement therapy, Johnson *et al.* (2007) found that women were 2 ½ times more tolerant of heart-attack mortality risk than of breast-cancer mortality risk. In a general-population survey of older adults, Hauber et al. (in press) found that respondents were willing to accept a risk of dying from stroke as high as 30% in return for a treatment that prevented Alzheimer's disease from progressing beyond the mild stage.

The possibility of a VSL cancer premium is worth additional study and the benzene case study could be improved by incorporating more discussion of this issue in light of recent research. The Council agrees that although these studies point to eventual VSL adjustments for cancer and other kinds of health risks, there is no scaling factor that policy makers can use today. Such adjustments would presumably vary with latency, type of health condition, and possibly other factors. Differences in the duration and the pain and suffering related to the illness and treatment may partly explain differences in risk-specific VSL. A cancer premium presumably would include such morbidity values, since they are inseparable from the mortality risk and should in principle be incorporated in the willingness-to-pay data used to construct the VSL.

The Council recommends that the EPA evaluate the implicit cancer premium assumed by including a pre-mortality morbidity value. The report could indicate, or at least speculate on, how large the cancer premium might be based on estimates provided in the literature. Clearly this recognizes an additional source of uncertainty in the valuation, and that uncertainty should be clearly discussed.

6. ***<u>Analyses of Individuals in High-Exposure Environments</u>. We conducted three supplemental analyses of CAAA-related impacts to Houston residents anticipated to have higher than average benzene exposures due to their location:1) residents living in census tracts with high modeled exposures; 2) residents living near roadways; and 3) residents living in homes with attached garages. Please comment on the data and methodological choices for these analyses with specific reference to:***

- ***EPA's choices regarding the most useful high exposure scenarios to evaluate; and***
- ***EPA's overall approach to valuing risk reductions using VSL, which does not account specifically for individuals who may have a higher than average baseline mortality risk due to high exposures to multiple HAPs and (as stated above in the question on a possible cancer premium) does not apply adjustments to account for the characteristics of the HAP risks being reduced.***

The case study demonstrates that it is feasible to estimate reductions in health risk to subpopulations having higher-than-average benzene exposure, though the significance of the heterogeneity of effects is not well explained. Using a linear exposure-response function, the total risk is unaffected by the distribution of exposure changes in the population. The report would benefit from discussion of what "high exposure" means in the context of this case study, where the individual lifetime risk is less than about one-in-ten thousand.

The Council notes that the proportional differences in risk reduction for populations in high- and median-exposure environments appear rather modest: the estimated risk reduction for residents of the two census tracts with the highest exposure in each of three counties range between 72 and 98 percent, a factor of 1.1 to 1.5 larger than the average risk reduction across the three counties of 65 percent. Near-road effects are somewhat larger, averaging a factor of 1.5 larger than in the absence of near-road effects for the ten census tracts with the highest on-road-related benzene concentrations and reaching a factor of two for one tract. Effects of attached garages may be much larger: accounting for these might increase estimated benefits of the CAAA regulations by 20 to 100 percent. The Council suggests that improved data on the exposure effects of attached garages may be relatively easy to collect (though beyond the scope of the case study) and may have a significant effect on the estimated regulatory benefits of benzene and perhaps other air toxics with significant in-garage sources. Council members found the assumption that each vehicle idles for five minutes in the garage before and after each trip surprisingly large (p. E-4). If this assumption significantly affects the exposure estimates, it should be better supported and perhaps modified.

The Council notes that the risk calculations for the analysis of high-exposure subpopulations are qualitatively different from the calculations for the main analysis. While the main analysis uses a life-table approach in which risks depend on exposure over a defined time period on the order of ten years, the high-exposure analysis calculates lifetime cancer risk using an attributable-deaths approach assuming lifetime exposure at a constant level and that lifetime risk is proportional to lifetime exposure. It would be worth highlighting this distinction and comparing estimates from the two approaches for the total population.

The Council suggests that individuals living near sources that have relatively frequent upset conditions (i.e., transient high-emission episodes due to equipment failure or other causes) may constitute another high-exposure subpopulation. While the total emissions may be only a small percentage of the total population-wide exposure, the local residents

may be at much higher risk. The Council encourages EPA to consider whether a subpopulation of this type can be identified, and whether its risk is likely to substantially exceed that of the general population.

Another source of high exposure that is not considered in the case study includes indoor sources such as cigarette smoking and emissions from consumer products. These sources often cause greater benzene exposures than those from outdoor sources, as discussed in the reports of the RIOPA studies, including one conducted in Houston. While the CAAA imposed no controls on such indoor sources, the increased proportion of the residual benzene exposure risk attributable to them should be included in the report's discussion section to provide an overall public health context.

With regard to VSL, questions concerning whether and how the valuation of mortality risk should be adjusted to account for characteristics of the risk (e.g., cancer compared with workplace accident; degree of voluntariness and controllability) are discussed in response to question 5. With regard to the higher baseline risk for individuals exposed to multiple HAPs, the Council notes that in theory VSL should be larger for otherwise similar individuals with higher total mortality risk (the so-called "dead-anyway" effect; Pratt and Zeckhauser, 1996). However, this effect depends on the magnitude of total mortality risk, and the difference in total risk attributable to HAP exposure is unlikely to be quantitatively important (e.g., annual risks for highly exposed individuals in the case study are on the order of one per million while total annual mortality risk is on the order of one per thousand or more, depending on age).

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